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The NICHD Connection

October 2011

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EDITOR IN CHIEF
Shana R. Spindler, PhD
Shana.Spindler@gmail.com

LAYOUT & DESIGN
Nichole Jonas

CONTRIBUTORS
Claudia Gebert, PhD
Elena Makareeva, PhD
Payal Ray, PhD
Shana R. Spindler, PhD
NICHD FARE recipients

PHOTOGRAPHY
Jeremy Swan
Stock.XCHNG

Interesting Opportunity: Being an Undergraduate Biochemistry Course Instructor

By Elena Makareeva, PhD

Dr. Makareeva

Last fall quarter (September – December 2010), I taught a 300-level undergraduate biochemistry course to budding young scientists and doctors who were conducting their research at the NIH campus. The course covered the biochemistry of proteins, nucleic acids and carbohydrates, in addition to common methods for biomolecule purification and characterization, such as chromatography, spectroscopy, NMR, X-ray, etc. Not only did I enjoy the course because the majority of the material was related to my lab experience, but also because the students were fun!

My students had travelled from Colgate University, located in the village of Hamilton in upstate New York, to participate in biomedical research at NIH. In addition to the biochemistry course, they attended one other class per week, and they spent their remaining time in their labs. All of the students were very much engaged in the course; they openly shared their relevant lab experience, excitement about science, and frustration with current experiments. Three of the total of seven students were applying to a graduate school, and one was in the process of applying to a medical school.

Throughout my postdoctoral training, I have always remained open to new teaching opportunities. When I received an email from the NICHD Deputy Director of Liaison & Training, Brenda Hanning, requesting experienced teachers for an upper-division biochemistry course, I immediately responded with high enthusiasm. Because I had taken advantage of other teaching opportunities at NIH, my application had the required credentials. For instance, before this formal biochemistry course, I participated in the following activities:

- For three years (2007-2009), I served as an instructor in the NICHD team-taught course "Becoming an Effective Scientist: Tools for NICHD Postbaccalaureate Fellows." This opportunity required a two-week commitment from each instructor.
- For five weeks in 2009, I participated as a leader of a Summer Intern Journal Club, organized by the Office of Intramural Training and Education.

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Letter from the Editor

An interesting theme has emerged in this month's issue: teaching and speaking about science. Perhaps born from the excitement of job-hunting season, it seems that communicating science has been on everyone's mind lately. Naturally then—as this newsletter serves as both a resource *for* fellows as well as a reflection *of* fellows—*The NICHD Connection* has much to offer in line with talking about science!

In our **"Interesting Opportunities"** column, Dr. Elena Makareeva proves that great teaching opportunities do exist for NIH fellows. The teaching science topic is continued with **Dr. Claudia Gebert's review** of Dr. Todd Zakrajsek's amazing "Strategies for Teaching" Seminar. If you missed the "Speaking About Science" workshop last month, check out **Dr. Payal Ray's recap** highlighting the key points.

In preparation for the NIH Research Festival this month, many of the NICHD Fellows Award for Research Excellence (FARE) recipients collaborated with *The NICHD Connection* to talk about their own science in a way accessible to any fellow, no matter the fellow's background. To learn about what your award-winning fellows are up to, peruse their research on **page 5**.

Finally, we have started a new column, "Life Outside Lab," where fellows can post fun pictures of what they like to do during the few hours they're not a work! Have a look on **page 12**. Do you have a fun picture too?

We look forward to bringing you the most up-to-date information and resources during this all-important job-hunting season. Until next month, be well!

Your Editor In Chief,
Shana R. Spindler, PhD

Would you like to contribute to *The NICHD Connection*? Please contact Shana Spindler at **Shana.Spindler@gmail.com**.

Review of Dr. Todd Zakrajsek's "Strategies for Teaching" Seminar

By Claudia Gebert, PhD

Following a night with a mere five hours of sleep, I arrived at Dr. Todd Zakrajsek's daylong seminar, "Overcoming apathy and creating excitement in the classroom: strategies for teaching from the psychology of learning." I had my coffee in hand (a strategy that I developed to avoid falling asleep during class), but it soon became apparent that with Dr. Zakrajsek, the coffee was unnecessary.

How I wish I had encountered the privilege of studying with only one teacher like Dr. Zakrajsek during university—either undergraduate or graduate! His enthusiasm about teaching and exploring ways to help students learn was infectious. Experiencing Dr. Zakrajsek's teaching makes me want to take on the challenge of becoming a teacher like him.

What is the hallmark of a teacher who facilitates instead of lecturing and who motivates students to understand rather than memorize facts? There is a concept known as "Lecture less, learn more." Students who have the opportunity to come across this style of teaching first acquire the basic knowledge via homework, then in class, under the teacher's guidance; students apply the knowledge by discussing concepts. The ultimate goal is to have students

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Review of "Strategies for Teaching" Seminar

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actually understand the material and apply the newly acquired information rather than simply regurgitating the facts.

This type of learning-centered teaching requires talking with instead of talking to students. An equal communication between teacher and students can be achieved by building a community during the first class. While the teacher holds the authority in the classroom, students, too, are given a voice, and their expectations on how the subject matter should be taught are heard and taken into consideration.

The challenge when teaching in the learning-centered style is to keep students engaged and focused on the subject matter. One of the multiple strategies to make coming to class attractive and to enhance students' participation is to distribute the handouts only after class and/or to keep the information provided on handouts to a minimum. That being said, if you missed Dr. Zakrajsek's teaching seminar this time, don't ask for a copy of the handout, because its information will be incomplete. Instead, try to attend his seminar the next time he comes to NIH—it will be worth every minute of your time!

Interesting Opportunity: Being an Undergraduate Biochemistry Course Instructor

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- During 2009-2011, I was an instructor and coordinator of lab activities in Take Your Child to Work Day at NIH.

Even with previous teaching experience, teaching a formal course was a big commitment. The biochemistry course ran from the beginning of September until Christmas. I had two classes a week for 1.5 hours each, totaling 3 hours per week. Each class had a lecture, interactive session, and homework. To monitor students' understanding of the covered material, I used pre-class tests, and for formal evaluation I had students complete two quizzes, two homework assignments, a presentation in the format of a journal club, a presentation on an individual research project, and a final exam—all of which needed grading!

As you can imagine, bench work was not easy to manage with my teaching schedule. Fortunately, my supervisor, Dr. Sergey Leikin, was very supportive. He understood that teaching a formal class takes a lot of time. In addition to three hours a week of classes, I spent a lot of time reading, preparing lectures and tests, and grading students' work. So, my major responsibility in the lab was supervising and helping other students on their projects. It worked well!

I have heard many times that teaching takes a ton of time, is exhausting, but can be extremely fun. Teaching this formal course gave me a chance to truly understand what teaching is all about. Not to mention, this teaching experience on my CV likely played a role in getting phone interviews from universities that focus on teaching undergraduates when I was applying to academic positions last year.

If you want a teaching career, teaching a formal course while you are a postdoc is a great start!

Recap of the "Speaking about Science" Workshop with Scott Morgan

By Payal Ray, PhD

As scientists, talking about our work is exciting and comes naturally to us. However, the art of clearly communicating scientific research to everyone in the audience can be challenging. To help NICHD trainees perfect their communication skills, the training office recently organized a "Speaking about Science" workshop, led by Scott Morgan, a communications coach who regularly helps NIH fellows. The workshop introduced participants to a basic framework for scientific talks, which can be applied across all research areas. The highlights of the workshop are discussed here.

SETTING UP THE QUESTION

To ensure a great start to your presentation, have a short and engaging title. The audience will be interested in your talk if you share common ground with them. This is of utmost importance for job talks, as the hiring committee might have people from various fields. The presenter must quickly set up a main question with a clear focus that is appealing to all. To do so, use the following format to design your introductory slides:

1. The collective scientific issues you share with the audience are _____
2. Of these you work on _____ because _____
3. More specifically you focus on ___

DATA SLIDES

The easiest way to set up a good presentation is to have a single theme. A helpful tip to establish a distinct theme is to put the most important data on one slide (the speaker called it the "money slide") and then build your presentation around that information. For data slides, use the two minutes per slide rule to stay within time limits. Introduce each slide, identify the highlight of the slide, and summarize it. A smooth transition between slides shows that the speaker is in charge of the presentation material.

WRAPPING UP

At the end of the presentation, time for questions and answers is important. After all, you do not want to travel a thousand miles to give a talk and be escorted off the stage due to a lack of time! So, monitor yourself carefully and have a short summary slide with a strong take-home message that the audience can remember.

Last, but not least, there is no substitute for practice. Rehearse your talk as many times as it takes for you to be comfortable.

Scott Morgan lists these and other useful strategies for giving scientific talks in his book, "Speaking about science: a manual for creating clear presentations." A copy of the book can be found with Brenda Hanning.

NICHD Award-Winning Research—For Everyone

Several of the NICHD Fellows Award for Research Excellence (FARE) winners who will present at this year's NIH Research Festival (October 24-28) have collaborated with *The NICHD Connection* to bring you a blurb about their research—understandable to everyone, whether you are a biologist, physicist, or clinician! If something piques your curiosity, check out their talk or poster at the festival!
In no particular order, the award-winning research of this year's NICHD FARE recipients:

Criminal Activities of Bacteria Revealed – *Legionella* Grand Theft Auto

By Yang Chen, Graduate Exchange Student from Peking University, Beijing

Legionnaire's disease, a severe form of pneumonia, sends nearly 8,000-18,000 people to the hospital each year, according to the Centers for Disease Control. The bacteria *Legionella pneumophila* causes Legionnaires' disease by infecting alveolar macrophage, an important cell type that resides in the surface layer of the lung alveoli. The bacteria hijack intracellular vesicle transport (the mechanism used by the cell to shuttle proteins around) and establish a camouflaged compartment resembling host-cell rough endoplasmic reticulum (the cellular machinery used to make and process proteins) to conceal the bacteria's own replication.

Legionella perpetrates its criminal activities in several ways, one of which involves transiently exploiting the activity of a key regulator of protein transport, called Rab1. The bacteria produce another protein, called SidM, which takes the Rab1 protein hostage and locks it in an active conformation through a process called AMPylation. With Rab1 as its hostage, the bacteria can elicit control over protein transport in the cell.

Most recently, we have identified how the bacteria convert Rab1 back into a non-AMPylated state—a prerequisite for the efficient use of Rab1's talents. *Legionella* use SidD, the first known de-AMPylase among bacterial virulent factors.

By mutating single pieces—one at a time—of the SidD protein, we identified an activity domain at the beginning of SidD and a targeting domain at the end of the protein. These findings offer insight into the molecular mechanism of SidD-based activities. Without SidD, the Rab1 hostage cannot be fully utilized in *Legionella* infected cells, indicating the importance of SidD for *Legionella*'s transportation theft!

Does *Legionella* use reversible modifications of hostage proteins to manipulate protein transport activities for *Legionella*'s own benefits? Our verdict: guilty as charged!

Don't Judge a Protein by Its Size—The Story of YneM

By Xuefeng Yin, PhD

In the past, researchers had ignored proteins of less than 50 amino acids within organisms. They were excluded in initial genome annotation and missed in classical genetic and biochemical characterization. Recent studies, however, have revealed an increasing number of small proteins that are critical to the bacterial cell. Small proteins have diverse roles, acting as: signal transducers, metal and nucleic acid chaperones, stabilizing factors for larger

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NICHD FARE recipients
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complexes, and adaptors for protein degradation. The conserved 31-amino acid protein called YneM is a good example that even the little guy can play a critical role in the cell.

YneM, identified in the bacteria *E. coli*, sits in the cytoplasmic membrane where it connects magnesium and phosphate regulation and possibly modulates their transport. YneM is highly produced when the magnesium level is low. It associates with a phosphate transporter in the membrane and potentially promotes export of phosphate through this transporter, thereby causing a drop in phosphate level inside the cell. This may release magnesium from the phosphate bound form and make magnesium ions available for enzymes to use.

The role of YneM exemplifies the idea that small proteins can accumulate under specific conditions and quickly respond to harmful environments at a relatively low energy cost. Generating larger proteins in response to a stressful condition would use a lot of energy for the cell, so in this case, maybe smaller really is better!

The Mystery of Brain and Liver Dysfunction in Niemann-Pick disease type C
By Celine Cluzeau, PhD

Niemann-Pick disease type C (NPC) is an inherited disorder characterized by cholesterol and fat build-up in the cells of the liver, spleen, and brain. This build-up leads to liver disease, progressive neurodegeneration, and ultimately, lethality. The root problem of the disease resides with a mutation in one of the *NPC1* or *NPC2* genes, which renders the cell compartments responsible for breaking down cholesterol and fats unable to complete the task. To date, there is no FDA approved therapy for NPC. Despite intensive study, the mechanisms leading to both brain and liver dysfunctions are poorly understood.

All cells function according to the genes that are being used. To determine why cells in NPC do not function properly, we compared which genes were being turned up or down in a mutant mouse model that lacked all NPC1 production (and closely recapitulated the human disorder) to the gene expression of healthy mice.

Upon studying the gene expression levels every other week, spanning the full disease progression from the first week to the terminal disease time of 11 weeks, we identified 222 altered pathways, including metabolic processes, immune responses and developmental signaling pathways. Among the genes that were differentially expressed at all ages, nine genes in the cytochrome P450 (CYP) family—enzymes that are involved in the metabolism of drugs and other intracellular compounds—were turned down in the mutant mice. CYP enzyme downregulation is a significant pharmacogenetic finding, since impaired CYP activity may result in altered drug metabolism by NPC patients and thus require alternative medication dosing.

Our findings are supported by preliminary data in mouse model, feline model, and NPC patients' livers, where there is a reduction of CYP activity in vitro. We are currently investigating CYP activity in vivo in the mouse model. In parallel, we are also working on the altered genes and pathways we identified with two objectives: 1) identifying proteins that we could use as biomarkers for NPC, and 2) understanding the link between these altered pathways and NPC presentation, to discover potential drug targets.

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Early Warning System to Predict If Cancer Will Spread

By Saravana Murthy, PhD

Cancer is a leading cause of death worldwide. The World Health Organization estimates that cancer will contribute to over 11 million deaths by 2030. In an intriguing twist, the initial tumor is likely not the deadly culprit. Rather, fatal scenarios arise after the primary tumor spreads to vital organs in a process called metastasis.

Clinicians are vigorously searching for a metastasis warning system. Traditional diagnostics rely on morphological and histological analysis, which are not very accurate and fail to identify and/or distinguish high risk and low risk patients for metastasis. These misinterpretations could lead to treatment failure and cause early fatality. Genetic biomarkers, however, could significantly improve these predictions.

The biomarker CPE-delta-N may be an important tool in a clinician's diagnostic toolbox. CPE-delta-N predicts the likelihood of tumor metastasis with accuracy rates approaching 90%. Tumor samples from 180 men and women with liver cancer showed that a doubling in the amount of CPE-delta N in the tumor, as compared to the surrounding tissue, predicted that the cancer was more likely to return or spread within two years. Similar results were found with pheochromocytoma (tumor of the adrenal glands), paraganglioma (neuroendocrine tumor), colorectal carcinoma and thyroid cancer. In fact, CPE-delta-N is not only a predictive marker for metastasis, it's also a root of the problem. In mouse models, CPE-delta-N drives another key gene called *NEDD9* to induce tumor metastasis.

Validation of the CPE-delta-N biomarker in a larger cohort of patients will hopefully lead to a future in which a patient's CPE-delta N levels could be used to guide individualized cancer care.

Mathematical Modeling of Membrane Pit Formation

By Anand Banerjee, PhD

Cells use a special process, called endocytosis, to engulf external material and package it into a transportable vesicle. To protect the vesicle, the cell uses a protein, called clathrin, which forms a highly-structured protective protein lattice. Clathrin mediated endocytosis (CME) is of fundamental importance to organisms in many ways, including—but not limited to—providing nutrition, regulating cholesterol metabolism and responding to hormone signals.

CME begins with the assembly of specialized proteins, including clathrin, on the internal side of the cell membrane. The protein assembly aids in the formation of small membrane invaginations called clathrin-coated pits (CCPs). Experiments show that there is considerable variability in the dynamics of CCPs. Some CCPs

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exist for only a few seconds and then disassemble abruptly. Other CCPs are relatively long-lived and grow steadily in size to form a vesicle. Even among the long-lived ones, there is a large variance in size and lifetimes. The origin of this heterogeneity is not clearly understood.

I am currently working on the development of a mathematical model to describe CCP assembly. My aim is to estimate the extent to which the stochastic nature of protein assembly is responsible for the observed variation in CCP dynamics.

Examining an Interneuron's Journey
By Brian Erkkila, PhD

Information processing by the brain and the rest of the central nervous system (CNS) is finely tuned by the interplay of excitatory and inhibitory neurons. Within the CNS, the hippocampus is a structure responsible for learning and memory, and it is no surprise that this region is of considerable importance in the pathologies of epilepsy, schizophrenia and Alzheimer's disease. The inhibitory neurons, or interneurons, of this region are not generated in their final locations in the neural circuit, but rather in a distinct brain region known as the ganglion eminence. During embryonic development, these interneurons migrate by "crawling" through the cortex until they ultimately reach their final destination in the hippocampus. Details about interneuron relocation have yet to be described.

We used genetically engineered mice that have a population of these interneurons labeled with a green fluorescent protein to track interneuron migration. Early in development, on embryonic day 12 (E12), the journey to the hippocampus takes up to four days. Closer to birth at E16, however, the journey takes only two days—despite the fact that the embryonic animal's brain has grown considerably and that the interneuron has had further to travel. In addition, we found that there was a significant reduction in the number of interneurons after birth. In fact, between birth and postnatal day 10, there was an ~80% reduction in the number of interneurons found in the hippocampus. Our future studies will focus on the attractive and repulsive cues responsible for interneuron migration as well as the identities of the interneurons lost during the first days of life.

How to Make Frog Gut Stem Cells from Tadpoles—Really!
By Kenta Fujimoto, PhD

One hundred years ago, Dr. J. F. Gudernatsch made the remarkable discovery that a substance in the thyroid gland could cause tadpoles to turn into frogs. Since Gudernatsch and others established that thyroid hormone (T3) is a developmental signal that triggers the onset of metamorphosis, scientists have used amphibians for studying the mechanism of T3 action and environmental toxicology.

T3 plays a major role in remodeling and or-

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ganogenesis during postembryonic development, a period around birth in mammals when T3 levels are high. Amphibian metamorphosis resembles mammalian postembryonic development, giving us a unique opportunity to study T3 function during development.

During amphibian metamorphosis, the tadpole intestine (predominantly a monolayer of larval epithelium, or a densely packed continuous sheet of cells) exhibits a dramatic transformation. The larval epithelial cells undergo cell death, and concurrently adult epithelial stem/progenitor cells (unique cells that can divide and replace damaged intestinal tissue) develop de novo through an unknown mechanism. How epithelial cells in tadpoles are slated to become adult stem cells is an active area of investigation.

We have shown that the expression of a gene called T3 receptor-coactivator protein arginine methyltransferase 1 (PRMT1) is increased in the small number of tadpole epithelial cells that appear to give rise to the adult stem cells. We have analyzed how PRMT1 is specifically upregulated in the cells fated to become stem cells. Our findings suggest the involvement of the c-MYC transcription factor, a protein responsible for guiding which genes in the DNA are to be used. Interestingly, c-Myc is turned up during metamorphosis prior to the increase of PRMT1 in the intestine.

From tadpole to frog, we have used amphibian metamorphosis to show that stem cell specific expression of PRMT1, potentially affected by the activities of c-Myc, may be an important mechanism during adult intestinal stem cell development.

Manganese Meets Its "Match"
By Lauren Waters, PhD

Cells are complex and crowded systems, full of proteins, lipids, DNA, RNA, and many other molecules. Mediating the correct binding of any two molecules in such a crowded environment is a daunting task for a cell. The problem is even more challenging when discriminating between two similar substrates, such as between different metal ions. To ensure that the correct metal is inserted into important enzymes, cells are thought to employ dedicated protein carriers called metallochaperones. These proteins "match" metal-using enzymes with the right metal, thereby guaranteeing that the enzymes are active and fully functional. However, only a few such metallochaperones are known to date, and only for a handful of metals.

We have recently identified a new candidate metallochaperone for the poorly studied but essential metal manganese in the model bacterium *E. coli*. Manganese promotes survival during oxidative stress and is required for bacterial virulence during pathogenesis. We discovered a previously unknown small protein of only 42 amino acids that is regulated by man-

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ganese. Elevated levels of this small protein, called MntS, caused a growth defect in the presence of manganese, but not any other metals. We also found that MntS could bind to manganese and other proteins, leading us to hypothesize that MntS may be the elusive manganese chaperone.

In vivo studies are underway to test the function of known manganese-using enzymes, such as manganese superoxide dismutase (Mn-SOD), in the presence or absence of MntS. In addition, we have purified MntS and are performing *in vitro* biochemical studies to elucidate mechanism of action of MntS. Did manganese finally meet its match in the lab? Our work to answer this question will advance our knowledge of manganese homeostasis and the intracellular trafficking of this important metal.

The Building Blocks of Neuron Communication—It's Not Babies' Play
By Madhav Sukumaran, PhD

Neurons communicate at junctions, called synapses, where chemical messages bind to specialized receptor proteins on the receiving neuron's membrane, thereby initiating an electrical response. One receptor subtype (the AMPA-type glutamate receptor) is responsible for fast, point-to-point communication between neurons. AMPA receptors are comprised of a number of subunits that can join together into different combinations, like Lego building blocks. These arrangements can influence the electrical properties of the neural connections and affect the function of the neuronal networks in which these neurons are embedded. The wrong configuration of AMPA receptor building blocks can contribute to the pathophysiology of such disorders as epilepsy, Alzheimer's disease, and stroke. A complete understanding of the molecular mechanisms underlying AMPA receptor assembly is a major goal of this work.

Using high-resolution biophysical, electrophysiological, and crystallographic assays, we investigated the role of the AMPA receptor N-terminal domain (NTD), a section of the protein that was previously implicated in receptor assembly. We showed that the NTD plays an organizing role in the initial assembly steps and identified which NTD components controlled the assembly process, much like finding hidden marks on Legos that would indicate two pieces should fit together.

Not all AMPA receptor assemblies are created equal, so we explored the physiological and pathological conditions that lead to the formation of favorable and unfavorable receptor populations. Our investigations of the NTD molecular architecture revealed that the N-terminal domain may be more dynamic than previously thought and may have an additional role in the neuron: directly regulating receptor electrical function through potentially novel mechanisms. As the title suggests, these blocks are not for babies.

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Cell Biology Is Like a Rock Concert

By Silviya P. Zustiak, PhD

Imagine a crowded rock concert. You are directly in front of the stage, but your cute date is stuck behind an enormous mass of moving people. As your date pushes and squeezes through the crowd, you worry about other attractive fans stealing your date's attention. Will your date make it through all those packed people? Believe it or not, this is an important question in cellular biology!

The typical cell is crowded with both charged and neutral molecules that slow down or completely prevent passive movement, called diffusion, of soluble proteins through the cell (like your date moving through people at a rock concert). This crowding effect is implicated in all cellular processes. For example, crowding has been shown to trigger aggregation of a special class of proteins that are associated with neurotoxicity in the neurodegenerative disorder Alzheimer's disease.

Despite vast interest in the subject, such hindered protein diffusion in the cell is still poorly understood. In other words, did your date fail to make it to the stage because there were too many people, or because your date found a more attractive person along the way?

In this project, we developed an in vitro cell model that has tunable binding and crowding to elucidate their relative roles on hindered protein diffusion. To simulate protein movement through a crowded cell, we labeled a small charged protein, called Ribonuclease A (RNase), with a fluorescent tag and monitored RNase diffusion through solutions of various charges (to simulate binding events) and various concentrations of dextran, a sugar polymer (to simulate crowding events).

In agreement with existing data, we observed a 5-fold decrease in RNase diffusivity in the most concentrated dextran solution. In this scenario, we measured that binding accounted for more diffusion inhibition than crowding. Further analysis revealed that one hundred times more crowder than binder was needed to achieve equivalent reduction in RNase diffusion. However, the data also suggested that at even higher crowder concentrations, similar to the levels found in a cell, crowding would overpower the effect of binding.

So what's the bottom line? According to our analogy, if it's a very densely packed concert, your date might not reach you due to the sheer number of individuals in the room. Otherwise, your date is likely to just get distracted by other cute people!

Life Outside Lab: What Fellows Do When Not at the Bench

Welcome to our new column where fellows can share fun pictures of "life outside lab" with the NICHD community. If you have a picture that you'd like to submit to the newsletter, please email it to Shana Spindler at Shana.Spindler@gmail.com with a short caption in the body of the email. This month, we have an exhilarating submission by Dr. Jana M. Kainerstorfer. It's good to know that your hair can still stand on end even in the water!

Jana is a postdoctoral fellow in the Section on Analytical and Functional Biophotonics. Her research is focused on non-invasive optical imaging technologies for tissue characterization. When not working on technology development, she enjoys scuba diving. Her recent adventure was diving in the Bahamas with docents of wild living sharks, who were fed and luckily didn't show any interest in the divers.

A Delicious Way to Spend the Afternoon

On September 21, 2011, over 80 fellows enjoyed 21 pizzas generously provided by the NICHD Program Heads in honor of National Postdoc Appreciation Week. *The NICHD Connection* offers an enormous thank you to the NICHD Program Heads on behalf of all NICHD postdocs for supporting this event for a second year.

The pizza party was a great opportunity to meet the new NICHD postdocs who joined their labs within the past year. Check out their photos on this page!

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October Announcements

BRENDA HANNING IS NAMED DEPUTY DIRECTOR OF LIAISON & TRAINING

The NICHD Connection would like to congratulate Brenda Hanning on her new title, Deputy Director, Liaison & Training of the NICHD Division of Intramural Research. On behalf of all NICHD fellows, congratulations Brenda!

Brenda Hanning

The NICHD DIR will be searching for someone for the Office of Education, to work with Brenda who will stay on as Director. The applicant will be expected to maintain the Office of Education's high level of commitment to training and academic/career support for fellows. A competitive candidate will be someone with a great deal of energy and passion for working with fellows across the spectrum, from summer intern and post-bac through doctoral fellows.

THE NIH INTRAMURAL RESEARCH PROGRAM HAS A NEW ONLINE SITE

Until last month, the collection of NIH intramural research programs (IRP) lacked an integrated website. Many institutes have their own online presence, but there was never a central hub for all NIH intramural research. The new site features research advances, the people behind the programs, and new opportunities at NIH, among other interesting tidbits about the IRP. Check out this new site at **irp.nih.gov**.

DON'T FORGET TO ATTEND THE NICHD EXCHANGE

The upcoming NICHD Exchange is about "Getting the Message Out: From Social Media to Grassroots Campaigns." Join Triesta Fowler-Lee, Yvonne Maddox, Jeremy Swan, and Kate Winseck for this interesting look at how to successfully achieve public outreach in the sciences. The NICHD Exchange meeting will be held on Friday, October 7, 2-4 p.m. at 6100 Executive Blvd, 5th floor conference room.

ONE-ON-ONE PUBLIC SPEAKING COACHING WITH SCOTT MORGAN

Gain a competitive advantage in this challenging job market! If you would like to arrange a one-on-one coaching session with expert Scott Morgan to prepare for a job talk, chalk, talk, or just to practice answering those tough interview questions, then contact Brenda Hanning at **hanningb@mail.nih.gov** to schedule a time.

October Events

FRIDAY, OCTOBER 7, 2-4 PM

NICHD Exchange
Getting the Message Out: From Social Media to Grassroots Campaigns
6100 Executive Blvd
5th floor conference room
To register, please visit insider.nichd.nih.gov/director/exchange

WEDNESDAY, OCTOBER 12, 12 PM

NICHD Fellows Committee Meeting
In front of the ground floor coffee shop in Bldg. 10, in the CRC atrium

MONDAY OCTOBER 24 – FRIDAY OCTOBER 28

2011 NIH Research Festival
Natcher Building